A COMPARATIVE STUDY ON THE MODALITIES OF TREATMENT IN LIVER ABSCESS AND THEIR OUTCOMES

Dissertation submitted in partial fulfillment of regulation for the award of

M.S. Degree in General Surgery (Branch I)

The TamilNadu

Dr. M.G.R. Medical University

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CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr.P.KANNAN.** *and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The TamilNadu Dr. M.G.R.Medical University, Chennai.*

Date :

Professor and Chief of
Surgery Unit III

Date :

Professor & Head
Department of Surgery

Date :

Dean
Coimbatore Medical College
Coimbatore - 641 010

DECLARATION

I solemnly declare that the dissertation titled **"A COMPARATIVE STUDY ON THE MODALITIES OF TREATMENT IN LIVER ABSCESS AND THEIR OUTCOMES"** was done by me from June 2010 to November 2011 under the guidance and supervision of Professor **Dr. P. Swaminathan. M.S., D.O.,**

This dissertation is submitted to the Tamil Nadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Dr.P.KANNAN

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CONTENT

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INTRODUCTION

Liver abscess was being considered as a major illness since ancient days i.e. from the era of Hippocrates (1). Till today liver abscess remains a major surgical problem. Introduction of antibiotics and development of advances in bacteriology and diagnostic methods have improved the outcome.

The early diagnosis is required for earlier and effective therapy. This is easier nowadays by using high resolution USG and CT scan. These imaging studies and percutaneous drainage under radiographic guidance with the use of antibiotics and surgical drainage improve the survival**.**

The treatment of liver abscesses has evolved greatly since 1938 when Ochsner et al. (2) demonstrated the reduction in mortality associated with operative drainage for all liver abscesses. A completely extraperitoneal approach was later described to minimize intraabdominal contamination in the preantibiotic era; however, with the use of perioperative parenteral antibiotics, a transperitoneal approach can be safely used, although extraperitoneal drainage is preferred. Since McFadzean et al.(3) first published a series on treatment of pyogenic liver abscesses with needle aspiration and antibiotics alone in 1953, a shift has occurred toward nonoperative management of liver abscesses. A series by Berger and Osborne in 1982(4) demonstrated the treatment of 62 patients with Hepatic abscesses with antibiotic therapy and needle aspiration; the mortality rate was 4%.

One should emphasize that percutaneous aspiration and drainage are simply extensions of standard surgical principles. As Gerzof et al.(4) have pointed out, the

Routes of percutaneous drainage are similar to the minimally invasive surgical routes of drainage.

The principles involved are to minimize spillage of abscess contents and decrease hematogenous spread with the least insult to the patient.

Once the diagnosis of a single or multiple liver abscess is made, broad-spectrum parenteral antibiotics should be started.

OBJECTIVES

1. To determine the outcome of medical management in liver abscess

2. To determine the outcome of surgical drainage in liver abscess

3. To compare the outcomes of both modes of management.

REVIEW OF LITERATURE

Liver abscess is a worldwide problem since ancient days. Some of the historical aspects of the anatomy of liver, liver abscess and development of modalities of treatment as follows

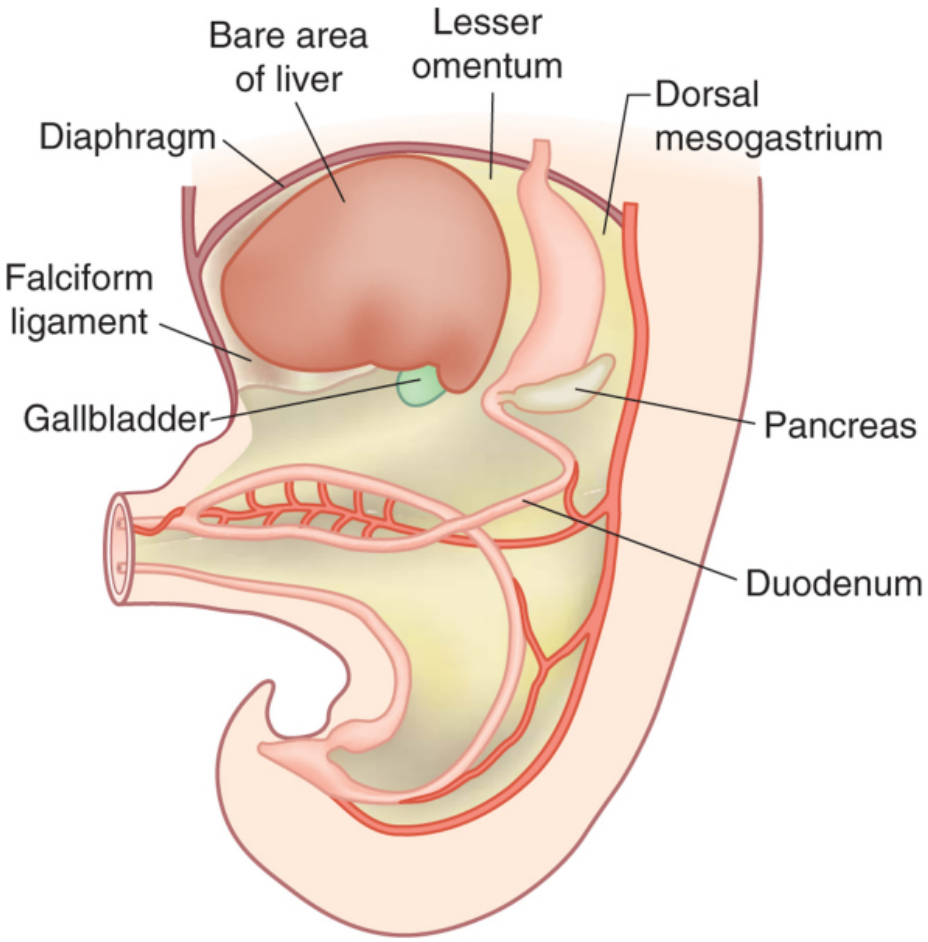
- 4000 B.C.- Hippocrates is credited with the first description of a hepatic abscess (1)
- 1654- Francis Glisson first physician to accurately describe the essential anatomy of the blood vessels of the liver(5)
- 1891- Councilman & Lafleur of Johns Hopkins Hospital coined the terms of amoebic dysentry & amoebic liver abscess(5)
- 1938- Ochsner & DeBakey in their classic paper on pyogenic liver abscess described 47 cases & reviewed the world literature (6).
- 1953- McFadzean & associates in Hongkong advocated closed aspiration & antibiotics for treatment of solitary liver abscess(3).
- 1957- Couinaud, Woodsmith & Goldburne described the segmental nature of the liver anatomy & opened the door even further to the modern era of liver surgery (5,7).

Normal Development & Embryology (8)

- The liver primordium is formed in the 3rd week of gestation as an overgrowth of endodermal epithelium.
- The connection between the hepatic diverticulum and the future duodenum narrows to form the bile duct and an out pouching of the bile duct forms the gall bladder and cystic duct.
- Hepatic cells develop cords and intermingle with the vitelline and umbilical veins to form hepatic sinusoids.
- Haemopoietic cells, kupffer cells and connective tissue form from the mesoderm of septum transversum (8).
- The mesoderm of the septum transversum connects the liver to the ventral abdominal wall and to the foregut.As the liver protrudes into the abdominal cavity these structures are stretched into thin membranes ultimately forming the falciform ligament and lesser omentum respectively.

FIGURE. 1

DEVELOPMENT OF LIVER



Anatomy

- A precise knowledge of the anatomy of the liver is an absolute pre-requisite to performing surgery on the liver and biliary tree.
- Liver is the largest visceral organ in the body and is primarily in the right hypochondrium and epigastric region, extending to the left hypochondrium (9).
- Liver is surrounded by a fibrous tissue known as Glisson's capsule (10).

Surfaces of the liver include:

A. A diaphragmatic surface in the anterior, superior and posterior directions.
B. Visceral surface in the inferior direction

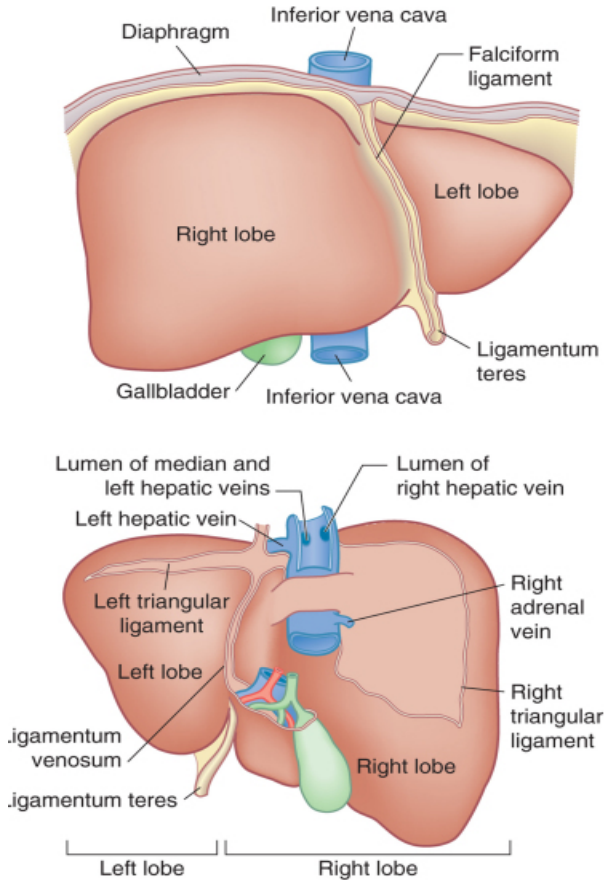
The porta hepatis serves as the point of entry into the liver for the hepatic arteries and portal vein and exit point for hepatic ducts (9).

Associated ligaments:

1. Falciform ligament
2. Hepatogastric ligament
3. Hepatoduodenal ligament
4. Right and left triangular ligaments
5. Anterior and posterior coronary ligaments.

FIGURE. 2

ANTERIOR AND POSTERIOINFERIOR SURFACES OF LIVER



Lobar anatomy:

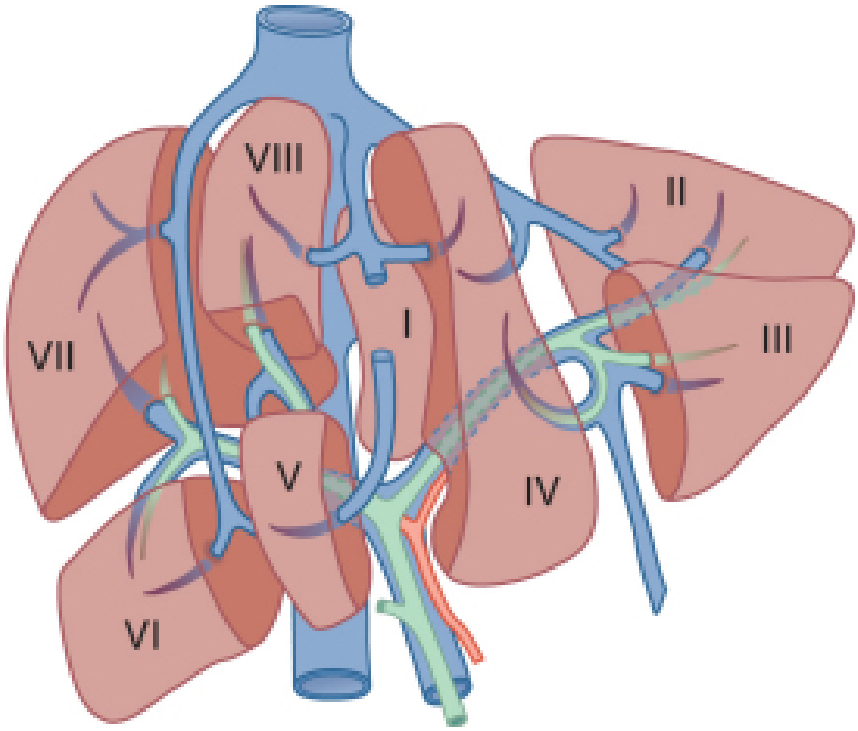
- Historically the liver was divided into the right and left lobes by falciform ligament. But anatomically incorrect in relationship to blood supply to the liver (11).
- A Cantlie's line running from the gall bladder to the left side of the inferior venacava-portal tissue divides the liver into right and left lobe (12).
- Right lobe is further divided into anterior and posterior segments.
- Left lobe is divided into a medial segment (quadrate lobe) and a lateral segment.

Functional anatomy:

- Functional anatomy of liver given by Woodsmith, Goldburne & Couinaud (7) is composed of 8 segments and each of which is supplied by single portal triad composed of portal vein, hepatic artery and bile duct (5).
- These segments are separated by the scissurae containing the 3 main hepatic veins and divide the liver into 4 sectors was highlighted by Bismuth (13).
- The main scissura (Cantlie's line) contains the middle hepatic vein and divide the liver into right & left hemi-liver.
- Right hemi liver is divided by right scissura (right hepatic vein) into anterior (V&VIII) and posterior segments (VI & VII).
- Left scissura (containing left hepatic vein) splits the left hemi liver into anterior (III & IV) and posterior (II) segments. Segment I called as caudate lobe (14) of the liver which is fully covered by lesser sac.
- The vascular inflow and biliary drainage to the caudate lobe comes from both right & left systems.
- Venous drainage of caudate lobe by multiple small veins drain posteriorly directly into the inferior venacava.

FIGURE. 3

SEGMENTAL ANATOMY OF LIVER



Blood supply:

Blood supply is by two ways:

1. Portal vein

The portal vein provides about 75% of the hepatic blood flow. The superior mesentric vein joins with the splenic vein posterior to the neck of pancreas to form the portal vein. At the hepatic hilum, the portal vein bifurcates into a larger right portal vein and a smaller left portal vein gives branches as in the figure.

2. Hepatic artery

This provides 25% of hepatic blood flow. The coeliac axis trifurcates into the splenic, left gastric, common hepatic arteries. Proper hepatic artery gives off right

and left hepatic arteries in the hilum. Left hepatic artery supplies the segments I, II, III. Middle hepatic artery is a branch of left hepatic artery and supplies IV segment. Right hepatic artery supplies the V, VI, VII, VIII segments.

FIGURE. 4

PORTAL VEIN AND ITS BRANCHES

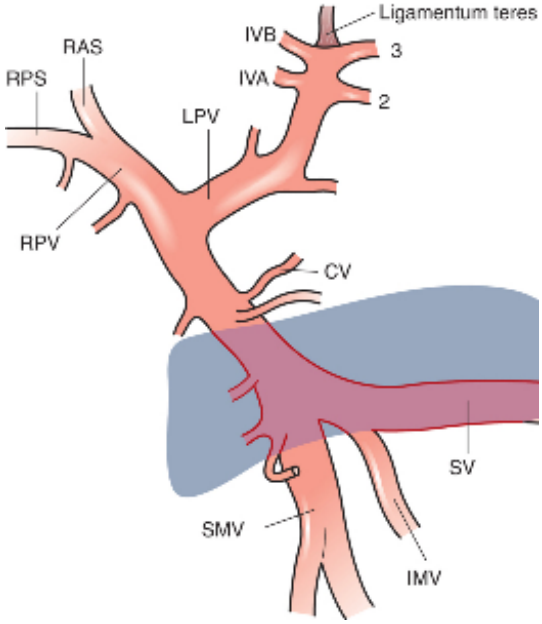
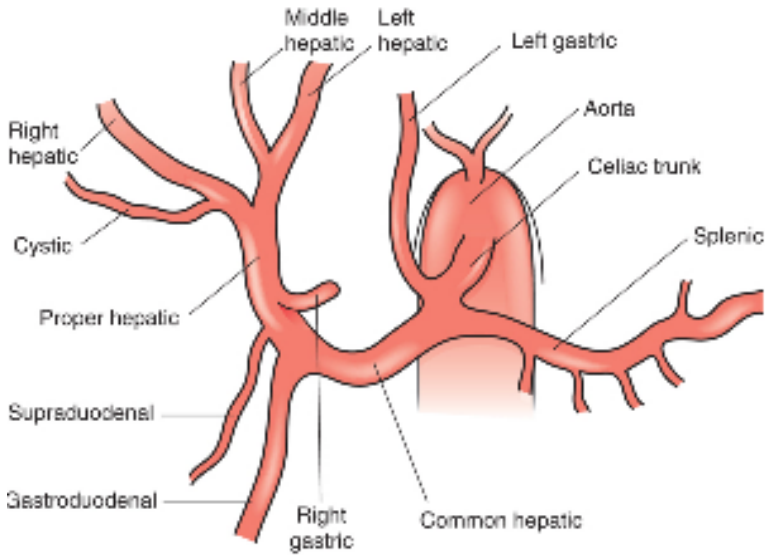


FIGURE 5

HEPATIC ARTERY AND ITS BRANCHES



3. Hepatic vein

The following veins drain the liver into the Inferior vena cava.

Right hepatic vein.(in the right scissura)

Middle hepatic vein (in the main scissura)

Left hepatic vein (in the left scissura)

Multiple small venous branches from the right posterior sector and caudate lobe drain posteriorly directly into the inferior venacava.

4. Bile duct

- Left hepatic duct drains segments ii, iii & iv.
- Right hepatic duct drains the right liver and is formed by the joining of the anterior sectoral duct (draining segments v&viii) and the posterior sectoral duct (draining segments vi & vii).
- Right & left hepatic duct joins to form the common hepatic duct. Cystic duct joins with the common hepatic duct and form the common bile duct.

LIVER ABSCESS

Kupffer cells act as a filter for clearance of micro-organisms. Abscess occurs when normal hepatic clearance mechanism fail or the system is overwhelmed. This leads to tissue invasion, neutrophil infiltration and formation of an organised abscess. Liver abscess is broadly categorised into:

1. **Pyogenic liver abscess**
2. **Amoebic liver abscess.**

Pyogenic liver abscess

The liver probably exposed to portal venous bacterial loads & clear this load normally.

When the inoculum of bacteria exceeds the liver's ability to clear it, leads to tissue invasion, neutrophil infiltration & formation of an organised abscess.

The potential routes of hepatic exposure to bacteria follow (15):

1. Biliary tree
2. Portal vein
3. Hepatic artery
4. Direct extension of a nearby focus of infection.
5. Trauma
6. Following hepatic artery embolisation & thermal ablative procedures.
7. Cryptogenic abscess.

1. Biliary tree:

Along with cryptogenic infections, infections from the biliary tree are presently the most common identifiable cause of hepatic abscess. Biliary obstruction results in bile stasis with the potential for subsequent bacterial colonization, infection, and ascension into the liver. This process is known as ascending suppurative cholangitis.

The nature of biliary obstruction is mostly related to stone disease or malignancy. In Asia, intrahepatic stones and cholangitis (recurrent pyogenic cholangitis are a common cause, whereas in the Western world (16), malignant obstruction is becoming a more predominant factor.

Other factors associated with increased risk include Caroli's disease, biliary *Ascaris* species infection, and biliary tract surgery. The common link between all causes of hepatic abscess from the biliary tree is obstruction and bacteria in the biliary tree.

Previous biliary-enteric anastomosis has also been associated with hepatic abscess formation, likely due to unimpeded exposure of the biliary tree to enteric organisms.

Forty percent of pyogenic liver abscesses of biliary origin are related to an underlying malignancy (17).

2. Portal vein:

Infectious disorder of gastro intestinal tract can result in an ascending portal vein infection (pyelophlebitis) with exposure of the liver to large amounts of bacteria.

Most common causes of pyelophlbitis are,

A. Diverticulitis
B. Appendicitis
C. Pancreatitis
D. Inflammatory bowel disease
E. Pelvic inflammatory disease
F. Perforated viscus
G. Colorectal malignancy, solid organ cancers as well as lymphoma and leukemia are present in 17-36% of patients with liver abscess (18).

3. Hepatic artery:

Any systemic infection (endocarditis, pneumonia, osteomyelitis) can result in bacteria and infection of liver through hepatic artery leads to multiple micro abscess.

Also associated with an altered immune response such as in patients with malignancy, acquired immunodeficiency syndrome (19,20), or disorders of granulocyte function.

4. Direct extension of a nearby focus of infection:

Like,

Suppurative cholecystitis

Sub phrenic abscess

Perinephric abscess

Perforation of the intestine

5. Trauma:

Penetrating or blunt trauma leads to intra hepatic haematoma and subsequently develop into an abscess.

Bacteria introduced from the trauma or affected area may be seeded from the systemic bacteria. Hepatic abscesses associated with trauma can present in a delayed fashion, upto weeks after the injury.

6. Following hepatic artery embolization and Thermal ablative procedure produce iatrogenic hepatic necrosis especially in patients with previous biliary enteric anastomosis.

7. Cryptogenic abscess: (15,21)

Cryptogenic abscess predominance in many series and are more common in recent case series. This may be due to,

1.Undiagnosed abdominal pathology

2.Resolved infective process at the time of presentation.

3.Host factors like diabetes, malignancy.

PATHOLOGY AND MICROBIOLOGY

A. LOBE INVOLVEMENT:

- In 75% of cases, right lobe of liver is commonly involved due to the preferential laminar blood flow to the right side.
- Left lobe is involved about 20%
- Caudate lobe involved in 5%.
- Bilobar involvement with multiple abscess is uncommon.

B. SIZE AND NUMBER:

- 50% hepatic abscess are solitary.
- The size of hepatic abscesses range from <1cm to several cms in diameter and can be multilocular or unilocular.

C.MODE OF BACTERIAL INFECTION:

- Abscess from pyelophlebitis & cholecystitis are polymicrobial(15,21,22) with high preponderence of gram negative rods.
- Systemic infection usually causes infection with single organism.
- Microbial negativity in the liver abscess are 50% (Ochsner's review,1938.As per 1990 report sterile abscess is about 10-20%)
- In the 1990, report sterile abscess is about 10-20%.
- Solitary abscess (usually right lobe) more likely to be polymicrobial and 40-60% by anaerobic organisms.

Organisms Isolated from Pyogenic Liver Abscesses(1)	
Category of Organism	**% of Patients**
Gram-Negative Aerobes	50–70
Escherichia coli	35–45
Klebsiella	18
Proteus	10
Enterobacter	15
Serratia	Rare
Morganella	Rare
Acinetobacter	Rare
Gram-Positive Aerobes	55
Stretococcal species	20
Enteroccocus faecalis	10
Beta-Streptococci	5
Alpha-Streptococci	5
Staphylococcal species	15
Anaerobes	40–50
Bacteroides species	24
Bacteroides fragilis	15
Fusobacterium	10
Peptostreptococcus	10
Clostridium	5
Actinomyces	Rare
Fungal	26
Sterile	7

Fungal & mycobacterial hepatic abscess (5):

- ✓ Rare
- ✓ Associated with immune suppression
- ✓ Following chemotherapy
- ✓ Multiple bilateral & miliary.

- Blood cultures are positive in 50% to 60% of cases
- Highly resistant organism in patients with indwelling biliary catheter causes multiple episodes of cholangitis.

Clinical features of liver abscess:

The clinical presentation of pyogenic liver abscess is usually subacute and non specific, leading to delays in presentation, diagnosis and treatment. In Seeto and Rocky's review (23) of 142 patients with pyogenic liver abscesses, the classic triad of fever, jaundice, and right upper quadrant tenderness was present in less than 10% of patients overall.

- Fever with chills
- Right upper quadrant pain
- Tenderness
- Diaphragmatic irritation causes pain in the right shoulder or right side of neck
- Chest findings 25% of the cases
- Hepatomegaly in 50% cases
- Uncommon signs are

  ascites

  splenomegaly

  severe sepsis.
- Fever is of hectic, "picket fence type", associated with chills and sweating.
- If the abscess ruptures, peritonitis and sepsis may be presenting features (22,23).

Investigations

- Leucocytosis (70-80% of cases).
- Liver function test—are always associated with underlying biliary disease. Alkaline phosphatase elevated in 80% of cases, Elevated bilirubin and transaminase in 50-67% patients.
- Anemia, hypoalbuminemia and prolonged prothrombin time are seen in 60-75% of patients (24).
- Total bilirubin increased in 20-50% patients.
- Mild elevation of prothombin time reflect a degree of chronicity.

None of the blood tests are specific to arrive at the diagnosis.

Radiology:

- Chest X-ray—elevated right hemi diaphragm, right pleural effusion or atelectasis.
- Occasionally these can be left sided findings in the case of an abscess in left lobe.
- Plain abdominal X-ray: shows air fluid levels or portal venous gas
- Ultrasonogram abdomen: round or oval area that is less echogenic than the liver, solid from cystic lesions. Sensitivity is 80-90%.
- CT scan demonstrates findings similar to ultrasound.
- High resolution CT scan can demonstrate very small abscess and can more easily pick up multiple small abscess. Sensitivity is 95 – 100%
- Barium enema to rule out occult diverticulitis.

Differential Diagnosis

Differentiating pyogenic abscess from other cystic infective diseases of the liver, such as amoebic abscess or echinococcal cyst, is important because of differences in treatment.

Pyogenic abscess is largely treated by antibiotics and drainage. Amoebic abscess is largely treated by antibiotics, and echinococcal cysts often require surgical management. Fortunately, echinococcal cysts can usually be diagnosed by history and characteristic radiologic findings .

The presentations of amoebic and pyogenic abscess, however, are more similar, with some notable exceptions that are critical in distinguishing the two. Amoebic abscesses generally occur in young Hispanic males in North America, whereas pyogenic abscess tends to occur in patients 50 to 60 years of age with no predominant gender or race.

Fever is common in both, but chills and symptoms of a severe acute bacteremia are more common in pyogenic abscess.

Serologic tests for *Entamoeba histolytica* antibodies are nearly always positive in patients with amoebic abscesses but are uncommon in those with pyogenic abscess.

A recent study comparing 471 patients with amoebic abscess to 106 patients with pyogenic abscess found age over 50 years, pulmonary findings on physical exam, multiple abscesses, and low amoebic serology titers to be independently predictive of pyogenic abscess (25).

Occasionally, differentiating the two is not possible, and diagnostic aspiration or a trial of antiamoebic antibiotics may be necessary. Unfortunately, aspiration is only diagnostic in amoebic abscess about 10% to 20% of the time (26).

Treatment of Pyogenic liver abscess

1. **Diagnosis**
2. **Drug therapy**
3. **Drainage**

Modalities:

- Parenteral antibiotic alone
- Percutaneous needle aspiration (single or repeated) + antibiotics
- Percutaneous catheter drainage + antibiotics
- Laparoscopic drainage + antibiotics
- Laparotomy-intra operative drainage + antibiotics
- Treatment of underlying cause
- Management of complications

General considerations:

- Resuscitation of shock
- Anti-pyretics/analgesics
- Correction of coagulopathy/Vitamin-K prophylaxis
- Antibiotic selection:

  Ampicillin + aminoglycoside + metronidazole (27)

  (to cover gram negative & anaerobic organisms)

  Or

  Cephalosporin (3^{rd} generation) + metronidazole (28)

Duration: >2 weeks to 4 months

- Co-morbid conditions: e.g., diabetic management, anti-hypertensives etc.,
- Anaemia
- Hypoproteinemia
- Anti-fungal therapy: Amphotericin-B

Parenteral antibiotics (4)

A trial of antibiotics alone should be reserved for patients with multiple small abscesses, low risk of abscess rupture, and lack of toxemia (i.e., no hemodynamic instability, patient does not feel acutely ill, etc.).

Once a commitment has been made to the antibiotic regimen, clinical response is gauged by defervescence, fall in leukocytosis, and resolution of symptoms, and should be reassessed frequently. Imaging with ultrasonography or CT can be used to assess resolution of abscess(es).

Lack of improvement after a reasonable course (10 to 14days) indicates failure of treatment.

Oral antibiotics should be continued for at least 4 weeks after discontinuance of parenteral antibiotics.

Worsening of fever, leukocytosis, and symptoms at any time also indicates failure of treatment and immediately qualifies the patient for a more aggressive treatment regimen involving a drainage procedure.

Percutaneous needle aspiration with parenteral antibiotics (4)

The first-line treatment for most patients with a pyogenic liver abscess should be percutaneous aspiration and antibiotic therapy. Aspiration involves as complete as possible drainage of the abscess cavity, and no catheter is left within the cavity. The patient's symptoms normally improve immediately after aspiration. Aspirated fluid should be sent for aerobic and anaerobic cultures.

Clinical response is again measured by a fall in fever and leukocytosis, and symptomatic improvement. Aspiration may have to be repeated when follow-up imaging is performed.

A report from Italy by Giorgio et al. (29) found an average of 2.2 ultrasonographically guided needle aspirations (range, 1 to 4) in 115 patients with pyogenic hepatic abscesses with an overall success rate of 98% with no mortality.

A randomized controlled trial by Rajak et al (30) in 1998 compared percutaneous needle aspiration to catheter drainage and also found no major complications and no deaths. In this series, after aspiration, abscess cavities were lavaged with saline, and an intracavitary antibiotic injection (gentamicin or metronidazole) was given in addition to systemic antibiotics. The clinical importance of placing antibiotics within the abscess cavity requires further prospective evaluation.

As other reports have been published of mortality associated with aspiration and antibiotics alone, the threshold to advance to percutaneous catheter drainage should be low.

Indications to proceed to percutaneous drainage include persistence of sepsis or worsening of clinical features, or failure to improve after a reasonable time period (5 to 7 days).

Although results vary widely in the literature, the number of patients for whom needle aspiration and antibiotic therapy fail is 3% to 20%.

Percutaneous catheter drainage + parenteral antibiotics (4)

Percutaneous catheter drainage with ultrasonography or CT guidance is indicated for patients for whom aspiration fails and for whom percutaneous drainage is not contraindicated.

Contraindications include coagulopathy, the lack of a safe or appropriate access route, and multiple macroscopic abscesses. Of note, the visualization of septae within the abscess cavity on CT or ultrasonography is not a contraindication to catheter drainage, as these rarely represent separate localized abscesses.

Using a modified Seldinger technique, the catheter is placed into the abscess cavity and left to straight drain in a position as dependent as possible to facilitate drainage. Clark and Towbin discussed this technique in detail. The catheter is flushed one to three times daily with 25 mL of sterile saline solution depending on the viscosity of the aspirate. The patient is again monitored for clinical improvement and cessation of drainage from the abscess (the catheter is slowly removed as the cavity shrinks).

Sinograms must be performed if drainage persists or if an enteric or biliary fistula is suspected; otherwise they are not routinely required. In 10% to 15% of cases, percutaneous drainage fails and intraoperative drainage is required.

Laparotomy-intra operative drainage + parenteral antibiotics (4)

Operative drainage of pyogenic hepatic abscesses is indicated for the following patients:

(a) patients who require laparotomy for the underlying problem,

(b) those in whom percutaneous catheter drainage fails, and

(c) patients with contraindications to percutaneous drainage.

(d) Patients whose liver abscesses rupture into the peritoneum also require laparotomy.

A midline or subcostal incision is performed, although the occasional patient benefits from a posterolateral eleventh rib approach. Intraoperative ultrasonography can be useful to help determine the ideal site for abscess drainage as well as identify portal structures and hepatic veins. Needle aspiration is used to localize the abscess precisely and can be used to identify the portal structures and hepatic veins.

The hepatotomy is then performed with electrocautery to open the abscess cavity. Drains (preferably closed suction) should be placed into the abscess cavity or cavities and exited via a separate abdominal stab wound. Laparoscopic approaches are reported to be successful. If the drainage does not contain bile, the drains can be removed reasonably quickly.

Laparoscopic drainage + parenteral antibiotics

- ❖ Limited use

Limitations:

- o Extensive perihepatic adhesions
- o Isolation of abscess is difficult
- o Laparoscopic techniques for biliary/pancreatic/colonic disorder

Resection of liver (5)

Indications:

- Recalcitrant abscess
- Hepatolithiasis
- Intra hepatic biliary stricture
- Infected hepatic malignancy

Underlying disease (4)

Modern medicine has created patients who have important underlying disease. The incidence of cryptogenic abscesses is increasing and is associated with cancer, diabetes, alcoholism, and cirrhosis. The most common underlying cause is biliary pathology, usually related to a benign or malignant obstruction.

Generally, malignant obstruction does not produce cholangitis or abscesses as the obstruction has developed in the presence of sterile bile.

Modern diagnostic and therapeutic techniques (ERCP, percutaneous cholangiogram, etc.), however, have led to increased manipulation, use of contrast material, and needle aspiration of the biliary tree, all of which contribute to the possibility of subsequent cholangitis.

Insertion of a percutaneous catheter or stent guarantees contamination and, if drainage is compromised, infection.

The relief of biliary obstruction or correction of stricture is imperative. This usually requires therapeutic ERCP and stents or open common duct exploration, clearance of stones if feasible, and T-tube drainage.

The use of choledochoscopy, extensive exploration and manipulation, or bypass depends on the patient's clinical status. In our enthusiasm to correct all defects, we should not lose sight of the objective: drainage and relief of obstruction.

Further endoscopic or operative therapy may be required after recovery from the acute process. These procedures may include sphincterotomy, biliary-enteric bypass, or resection of liver for ductal obstruction.

Abscess via direct extension is usually secondary to acute cholecystitis. Cholecystectomy is required with appropriate drainage and operative cholangiography to ensure that the common duct is not obstructed.

If portal vein dissemination, which is less common, is the problem, the focus (usually appendicitis, Crohn disease, or diverticulitis) must be managed; resection is best if possible.

Hepatic artery bacteremia requires the identification of the focus (e.g., total parenteral nutrition line, endocarditis, tuberculosis).

Hepatic trauma that leads to abscess may require evacuation of the hematoma or débridement of necrotic hepatic parenchyma, or both. Percutaneous drainage and time will usually lead to a good outcome. Failure to resolve the underlying process leads to persistence or recurrence of the infection.

Prognosis (4)

The prognosis of hepatic abscesses has improved dramatically with concurrent advances in diagnostic and treatment modalities. In the report by Huang et al.,

mortality for pyogenic hepatic abscesses was 65% from 1952 to 1972, and 31% from 1973 to 1993.

Sepsis and multiple-organ dysfunction syndrome are now the most common causes of death in patients with liver abscesses.

With modern imaging, antibiotics, and minimally invasive drainage, mortality for hepatic abscesses should be less than 10%.

In Asia, *Klebsiella* abscesses have been more common and in diabetics have been associated with increased mortality and serious metastatic disease, particularly endophthalmitis.

Amoebic liver abscess

Amoebic liver abscess is caused by the parasitic protozoan *Entamoeba histolytica* . In 1890 Sir William Osler described the first North American case when , after an attack of dysentery , while in Panama , a physician's stool and abscess fluid were both found to contain amoeba (1).

Councilman and Lafleur of Johns Hopkin's Hospital coined the term amoebic dysentery and amoebic liver abscess in 1899 (31).

Entamoeba histolytica (Schaudinn,1903)

The parasite causing diarrhoea & dysentery and liver abscess in men.

Lambl (1859) first discovered the parasite. Losch (1875) proved its pathogenic nature , while Schaudinn (1903) differentiated pathogenic and non-pathogenic types of amoebae.

***Incidence*:**

The vast majority of these infections are acquired in the developing world. High risk groups in the United States include sexually active homosexual men,immigrants, tourists who travel to endemic areas, institutionalized people,and those with HIV (32). Amoebiasis follows a bimodal age distribution. Those living in developing countries have a greater risk and an earlier age of infection. Low socioeconomic status and unsanitary conditions are significant independent risk factors for infection. Ten times common in men and is a rare in children.

Life cycle of the E. Histolytica

Life cycle is as shown in the picture, involves cysts invasive trophozoites and focally contaminated food or water to initiate the infection (33,34).

Reservoir of infection : Human

Mode of infection : Faeco-oral

Role of carriers : Handling of food by infected individuals (cyst passers or cyst carriers) appear to be a very common method.

Two types of carriers

1.Contact

2.Convalescent

***Stages*:**

1.Cystic stage

2.Pre cystic stage

3.Trophozoite stage

Trophozoites are the infectious form, which exist in the human gut to form cysts, which colonizes in the gut wall. Cystic stage is dangerous to the community as they are responsible for the carrier stage.

FIGURE. 6.

LIFE CYCLE OF E.HISTOLYTICA

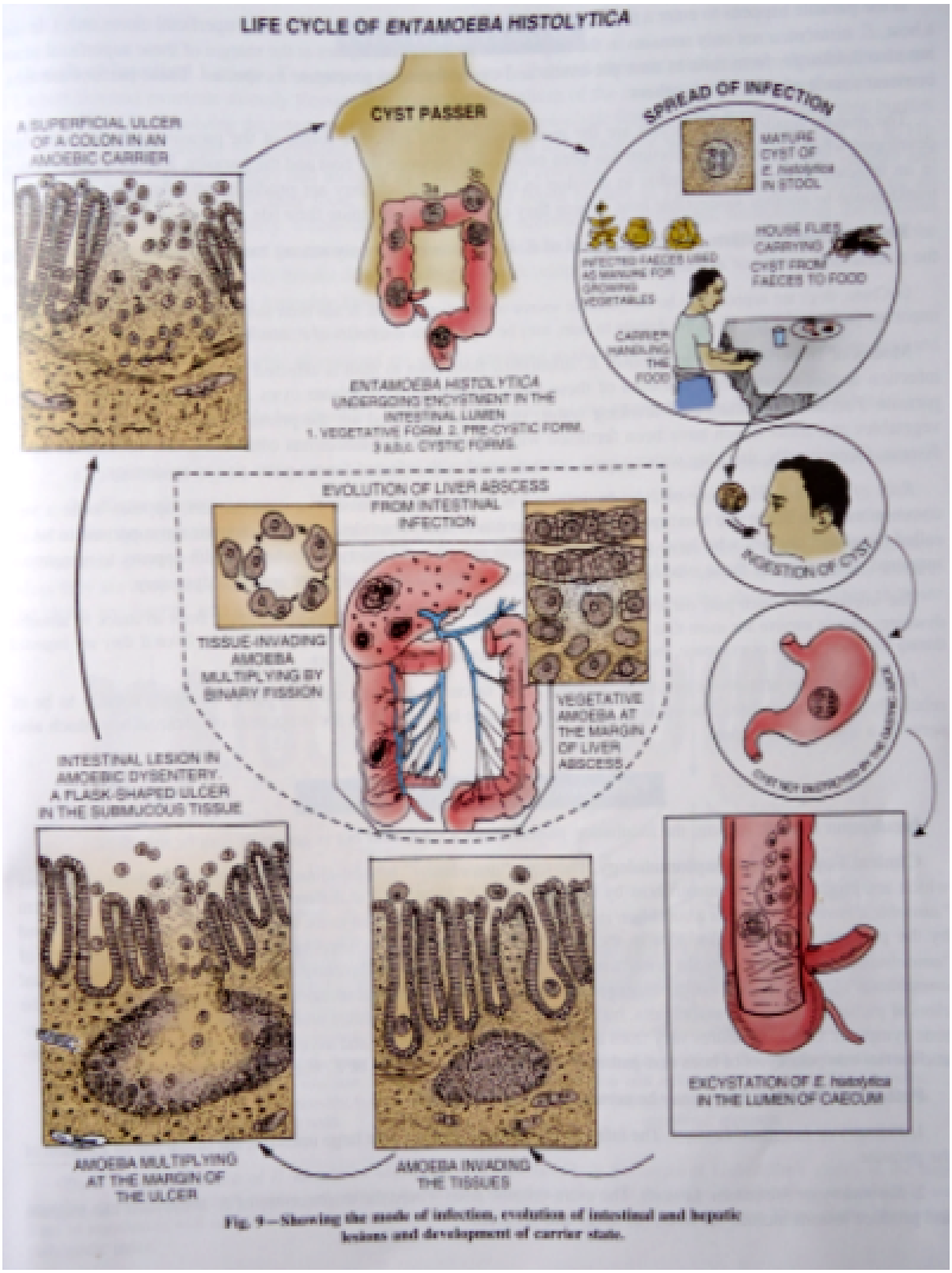


Fig. 9—Showing the mode of infection, evolution of intestinal and hepatic lesions and development of carrier state.

Pathogenesis of amoebic liver abscess

Once ingested, the cyst are not degraded in the stomach, and pass to the intestines, where the trophozoites are the released and passed on to the colon.

In the colon, the trophozoite can invade the mucosa, resulting in disease. The trophozoite reach the liver through the portal venous system, usually upper and posterior portion of the right lobe are affected. It has the capacity to lyse tissues through a complex set of events, including cell adherence, cell activation and subsequent release of multiple enzyme resulting in necrosis by enzymatic cellular hydrolysis which leads to progressing localized hepatic necrosis resulting in a cavity containing acellular proteinaceous debris surrounded by a rim of invasive amoebic trophozoites. In early stage abscess is associated with an accumulation of poly-morphonuclear leucocytes which are then lysed by the trophozoites (5).

Tissue invasion is probably influenced by host's diet(cholesterol is believed to be a nutritional factor for E. histolytica and is also thought to increase its virulence),special intestinal flora and enzymatic proteolytic activity of the parasite itself

- In early stage amoeba will be found in the abscess.
- In later stages, amoeba are not easily to be detected.

FIGURE. 7

MACROSCOPIC APPEARANCE OF AMOEBIC LIVER ABSCESS

Pathology:

To the naked eye the appearance of abscess area is reddish brown in colour with a semi fluid or grumous consistency **(35).**

Hepatic amoebic abscess is the result of liquefactive nerosis of liver producing a cavity full of blood and liquefied liver tissue, as reddish brown chocolate coloured anchovy sauce pus which is odourless, unless secondary bacterial infection has taken place. Progressive hepatic necrosis continues until Glisson's capsule is reached, because the capsule is resistant to hydrolysis by the amoebae and thus the amoebic abscess tend to abut the liver capsule. The cavity is typically crisscrossed by portal triads protected by this peritoneal sheath early on, the formed cavity is ill defined with no real fibrous response around the edges, but a chronic abscess can develop a fibrous capsule and may even calcify. Like pyogenic abscess , amoebic abscesses tend to occur mainly in the right lobe of liver (5).

Microscopic pathology:

There are 3 zones which can be differentiated from the centre to the periphery.

- The central zone of cytolysed granular material with no amoebae.
- An intermediate zone consisting of degenerated liver cells, a few leucocytes, connective tissue cells, red blood cells and an occasional trophozoite of E.histolytica
- A peripheral zone consisting of congested capillaries with varying degrees of necrosis of liver cells. The amoebae can be seen to be multiplying in this area and invading the adjoining healthy liver tissue (35).

Clinical features

About 80% of patients come with symptoms lasting from a few days to few weeks.

- Fever with chills
- Anorexia
- Right upper quadrant pain and tenderness
- Hepatomegaly
- Diarrhoea in 25% of patients
- Active amoebic colitis in 1/3 of patients
- Jaundice when compressing the biliary tree
- Weight loss

- Myalgia
- Sympathetic pleural effusion
- Pleuritic or shoulder pain
- Lung signs
- Peritonitis- if ruptured into the peritoneum
- In rare cases it may rupture into pleural space , pericardium and other intra abdominal organs and produce symptoms & signs

Laboratory results:

- Leucocytosis without eosinophilia
- Anaemia
- Mild abnormalities in liver function tests
- Elevated prothrombin time
- Circulating anti-amoebic antibodies-Serum antibodies are positive in 99% of patients with liver abscess (36).
- Indirect haemagglutination test has a sensitivity of 90%.
- Enzyme immune assay has a sensitivity of 99%.
- Chest radiographs are abnormal in two-thirds of patients with amoebic liver abscess and frequently show pleural effusion infiltrates or an elevated hemidiaphragm.

- Ultrasound, CT scan are excellent methods of detecting amoebic abscesses but are nonspecific (37).

- In 75-80% of cases, only a single abscess is present and in the right lobe,10% are in the left lobe, and the rest are multiple. Six percent may present as a caudate lobe abscess.

- Resolution may be complete or result in a small cystic cavity that resembles a simple cyst of the liver (38).

- Diagnostic aspiration- anchovy sauce pus.

Differential diagnosis:

The differential diagnosis of an amoebic liver abscess can be broad and include such diseases as viral hepatitis, echinococcal disease, cholangitis, cholecystitis, or even other inflammatory abdominal disorders such as appendicitis. Malignant lesions of the liver can also have similar presentations in atypical situations. Occasionally, primary pulmonary disorders must be considered. In the main, the most important distinction to be made is between pyogenic and amoebic abscess (5).

Course and termination of amoebic liver abscess

It may heal up spontaneously leaving an encysted mass, the contents of which may be dried up fibrosed or even calcified (35).

Liver abscess may have the following terminations (35).

- A right sided liver abscess may rupture

- Externally → granuloma cutis
- Into the lungs→ haemoptysis- anchovy sauce sputum
- Into the pleural cavity→ empyema thoracis
- Below the diaphragm→ subphrenic abscess
- Into the peritoneal cavity→generalised peritonitis

FIGURE. 8

COURSE & TERMINATION AMOEBIC LIVER ABSCESS

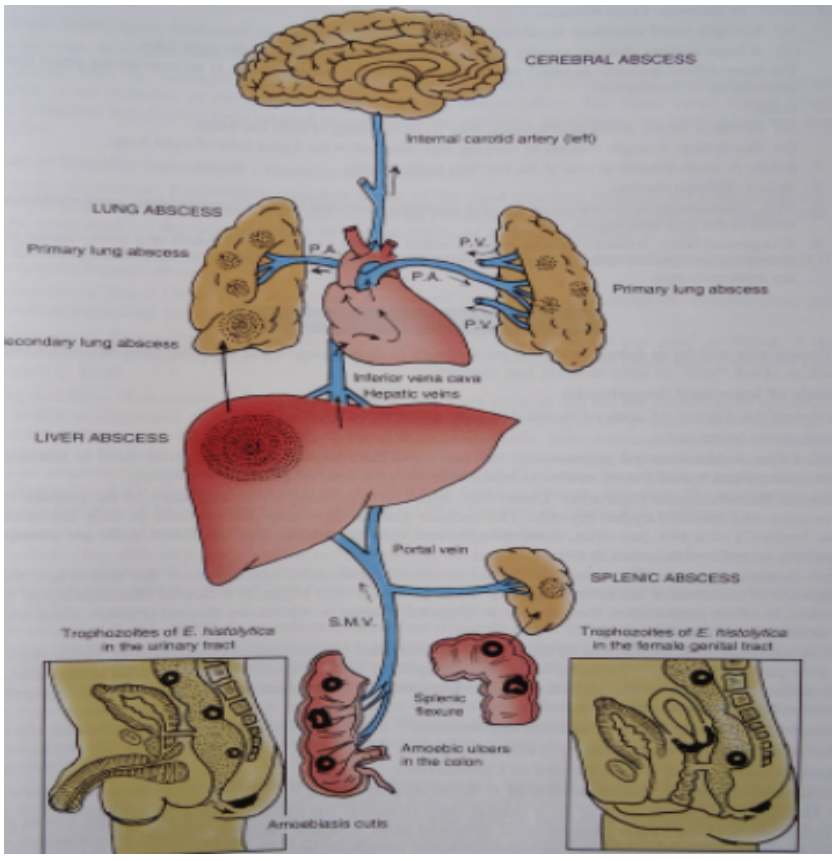
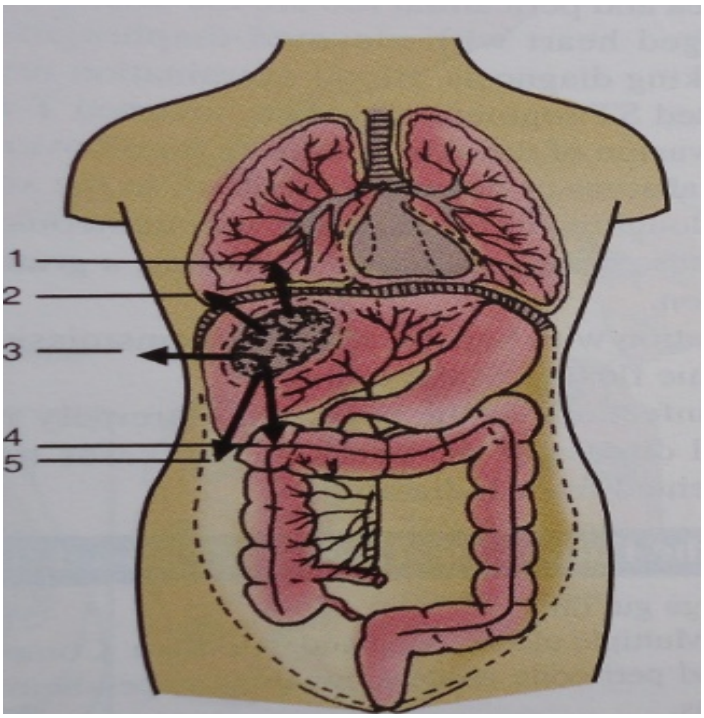


FIGURE. 9



1. Lung

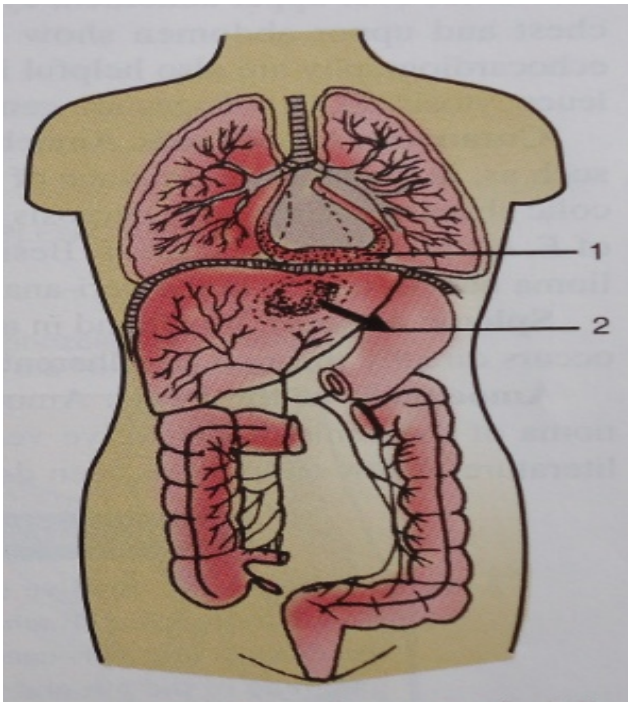
2. Pleura

3. Cutaneous

4. Colon

5. Peritoneum

Sites of rupture of a right & left sided liver abscess



1. Pericardium

2. Stomach

A left sided liver abscess may rupture into

- Stomach→ pus is vomited out- hemetemesis.
- Pericardium → purulent pericarditis with tamponade(33)- fatal
- Externally
- Left pleural cavity-empyema thoracis.

- A liver abscess situated on the interior surface may rupture into
  - Bowel(transverse colon or duodenum)→ diarrheoa and discharge of `pus` in the stool.
  - Peritoneal cavity→ fatal peritonitis
- A liver abscess situated on the posterior surface may rupture into: inferior vena cava→ fatal
- Other sites of rupture into
  - The CBD
  - Pelvis of the kidney
  - Perinephric tissue

Treatment of Amoebic liver abscess (5)

A.Antibiotics:

Imidazole (eg. IV metronidazole 500 mg tds 10-14 days -95% cure rate.

Clinical response within 3 days, At 5 days an 85% cure rate is achieved,and this response may be increased to 95% by 10 days. Five to fifteen percent of patients with amoebic liver abscess may be resistant to metronidazole (38).

Complete resolution of abscess in 3-30 days(according to initial size)

Emetine/dehydroemetine- for pulmonary complications very ill – multidrug therapy

Chloroquine with emetine- metronidazole resistant amoebic strains

A.Therapeutic aspiration:

Patients receiving parentral drug – symptoms persisting after 72 hrs.

- To rule out secondary bacterial infection
- Risk of rupture
- Large abscess > 5cms diameter
- Abscess volume > 250 ml
- Left lobe abscess
- Lesions with marked tenderness
- Diaphragmatic elevation
- Pregnancy (metronidazole contraindicated)

Bacterial coinfection of amoebic liver abscess has been observed; therefore addition of antibiotics,drainage,or a combination of both, to metronidazole therapy may be necessary (37).

A. Percutaneous drainage:

- Wide diameter catheter – high viscosity
- Indicated in pulmonary,peritoneal pericardial complications
- Surgical drainage done in case of

  failure of conservative therapy

  life threatening haemorrhage

  abscess rupture

  viscous perforation

  septicemia(secondary bacterial infection)
- Cavity opened, scooped out, irrigated with quinine hydrochloride (1:1000) solution.

OUTCOME:

The majority of patients with amoebic liver abscess defervesce within 3-4 days of treatment (39); however, left untreated amoebic abscesses are often fatal. Mortality rates of 0-18% are reported, with higher rates occurring secondary to delay in diagnosis,or when secondary bacterial infectin or complications occur. Independent risk factors for mortality include serum bilirubin> 3.5mg/dl, encephalopathy, hypoalbuminemia and multiple cavities (40).

MATERIALS AND METHODS

A prospective study over a period of 18 months from June 2010 and November 2011. This study was conducted at C.M.C.H, Coimbatore.

In this study, 50 patients were subjected to detailed history clinical examination, routine investigations, radiological investigations like USG abdomen and pelvis & Computerised Tomographic scan.

USG- abdomen was done in all patients. CT scan was done in patients with multiple liver abscesses, caudate lobe abscess, left lobe liver abscess and ruptured liver abscesses.

After investigations, they underwent different modalities of treatment:

1. Conservative management with antibiotics alone.
2. Percutaneous aspiration with antibiotics- USG guided.
3. Closed tube drainage with antibiotics- USG guided.
4. Open surgical drainage with antibiotics.

Immediately after admission all patients were put on the intravenous antibiotics, regardless of the organism or condition of the patient.

Depending upon the number and size of the cavity, nature of the illness, location of the abscess cavity, patients were categorized into the following methods.

1. Antibiotics alone- less than 200 ml

   The routine drugs were used like ampicillin + aminoglycosides+ metronidazole

   or

Third generation cephalosporins and metronidazole which were supplied by our hospital.

2. Patients with following criteria were taken for percutaneous drainage.
    - Patients who continued/ worsened to treatment with antibiotics alone.
    - Liver abscess with more than 5cm in size(200ml)
    - Clinical or USG features suggest impending rupture.
3. Open drainage was done in patients falling in Kapoor's criteria.
    - Thick pus which could not be aspirated.
    - Patients with ongoing sepsis ever after antibiotics treatment and percutaneous drainage
    - Multilobular abscess
    - Abscess in the left lobe
    - Ruptured abscess

Bacterial culture and sensitivity was done in the patients has undergone with aspiration and open surgical methods and the antibiotics were changed according to culture and sensitivity.

Radiologically guided aspiration and drainage was performed under local anaesthesia, after putting the patient on Vitamin K Prophylaxis for 3 days. After emptying the cavity 25 to 50ml of metronidazole was flushed through the same needle for an chovy sauce pus cavities.

Figure – 10
NEEDLE ASPIRATION

With an 18 gauze needle and with different size drainage catheter placed by Modified Seldinger technique. In this, the catheter was placed in the abscess cavity and left to straight drain in apposition as dependent as possible to facilitate drainage.

Figure – 11
LEFT LOBE LIVER ABSCESS

Figure – 12 & 13
TUBE DRAINAGE

The patient was again monitored for clinical improvement and cessation of drainage from the abscess(the catheter was slowly removed as the cavity shrinks). The pus aspirated at the time of drainage was routinely sent for microbial culture and sensitivity.

The recommended duration of parenteral antibiotic therapy is continued for 2-3 months or until there is a favourable clinical response.

Complementary oral antimicrobial therapy continued for further 2-4 weeks or until clinical, biochemical and radiological follow up demonstrates complete resolution of abscess cavity.

Inclusion criteria:

- Patients >18 years,
- Patients presenting with hepatic abscess

Exclusion criteria:

- Patients <18 years,
- Pregnancy.
- The data will then be analysed statistically.

RESULTS

The data was tabulated and analysed as follows :

Table 1 : MODE OF PRESENTATIONS

Sl.no	Involved part	No.of pts	%
1	Only right lobe	41	82
2	Only left lobe	4	8
3	Both lobes	4	8
4	Caudate	1	2

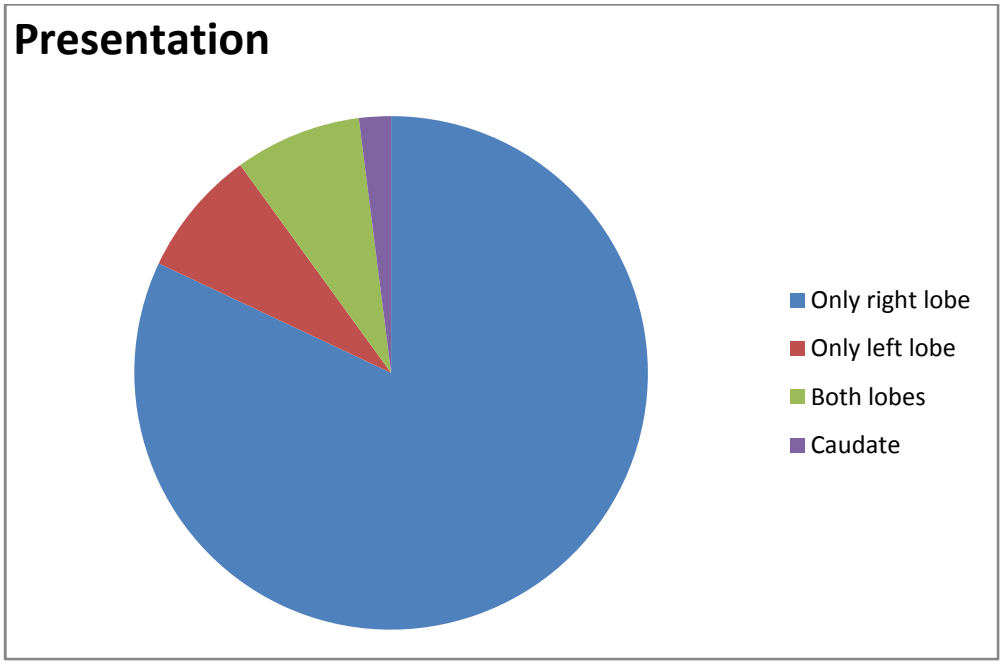


Table 2 : NUMBER OF ABSCESS CAVITY

S.no	No of cavity	Right	Left	Caudate	Both lobe
1	Single	36	4	1	0
2	Two	0	0	0	4
3	Multiple	5	0	0	0

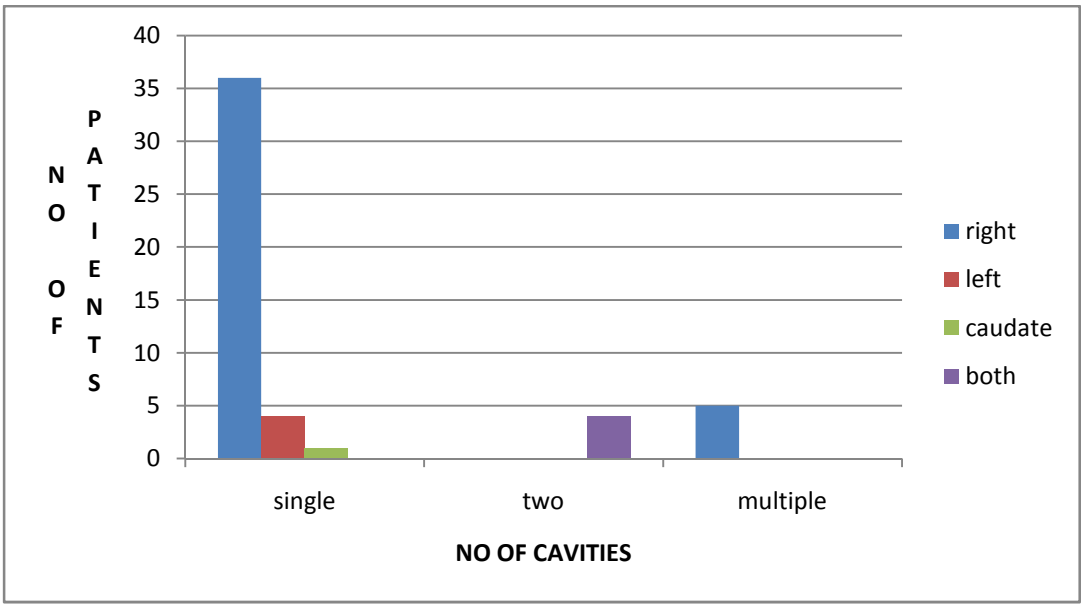


Table 3 : NUMBER OF CAVITY AND ITS MODE OF TREATMENT

S.No.	No.of cavity	I.V.Antibiotics Alone	Aspiration + antibiotics	Tube drainage+ Antibiotic	Laparotomy+ Drain+ Antibiotics
1	Multiple	5	3	0	0
2	Two	0	4	0	0
3	Single	12	26	4	3

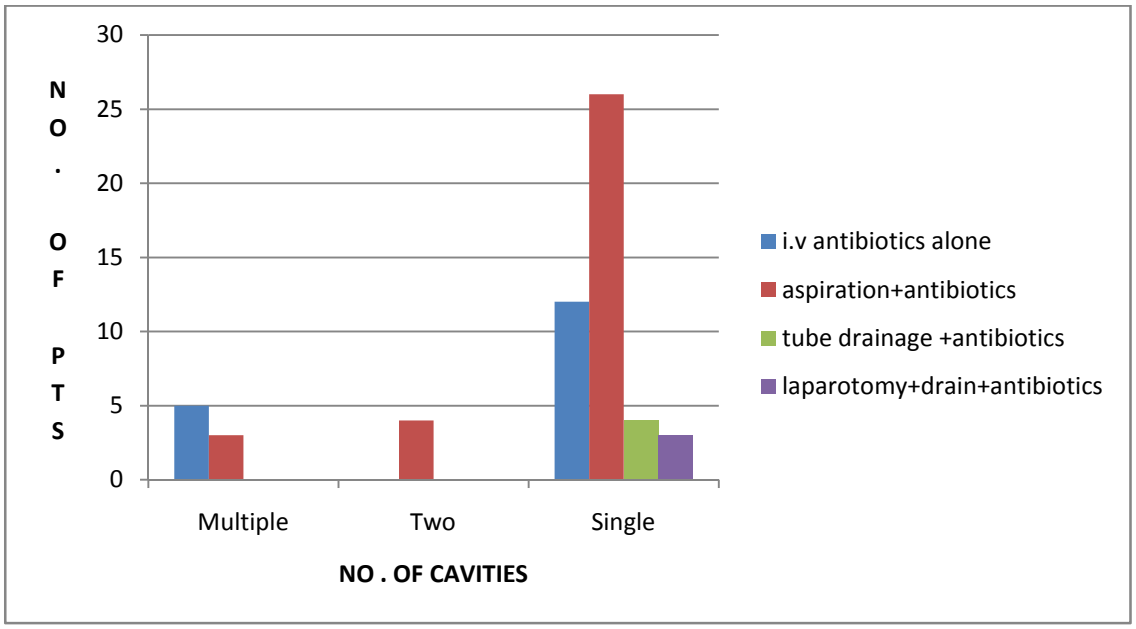


Table 4 : MODE OF OUTCOME WITH PARENTERAL ANTIBIOTICS ALONE

S.No.	Lobe	No.of patients	Cured	Failure
1	Right	15	10(66.6%)	5(33%)
2	Left	1	0	0(100%)
3	Both	0	0	0
4	Caudate (small abscess)	1	1(100%)	0

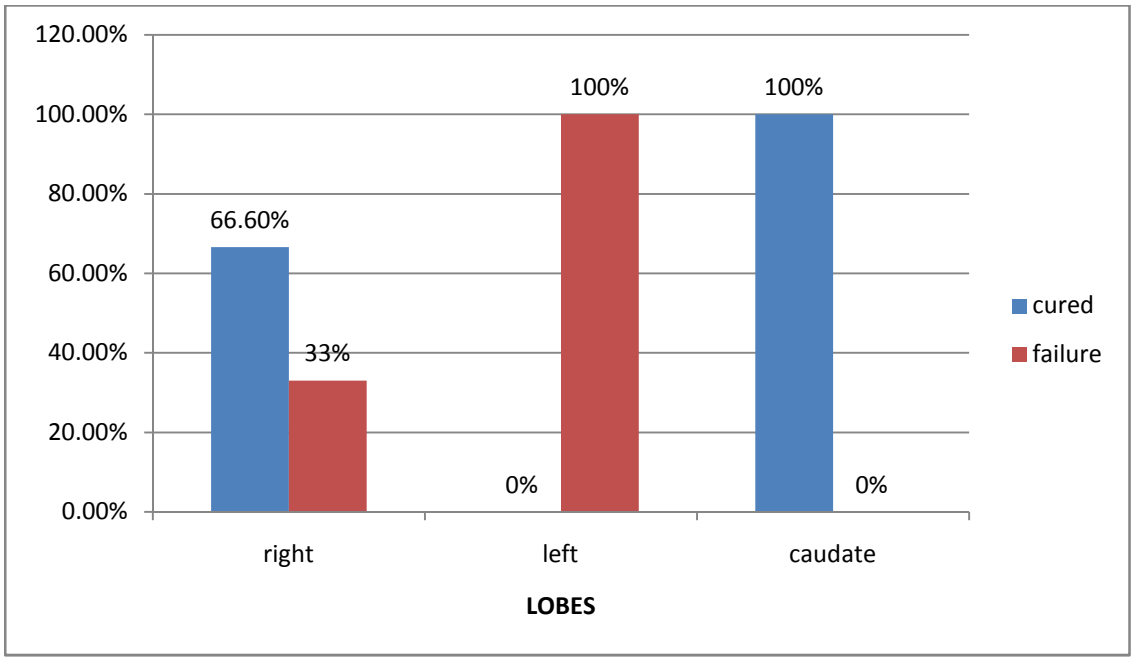


Table 5 : NUMBER OF ASPIRATIONS

S.No.	Lobe	No.of patients	1st aspiration	2nd aspiration	3rd aspiration
1	Right	27	27	9	4
2	Left	2	2	1	1
3	Both	4	4	4	2

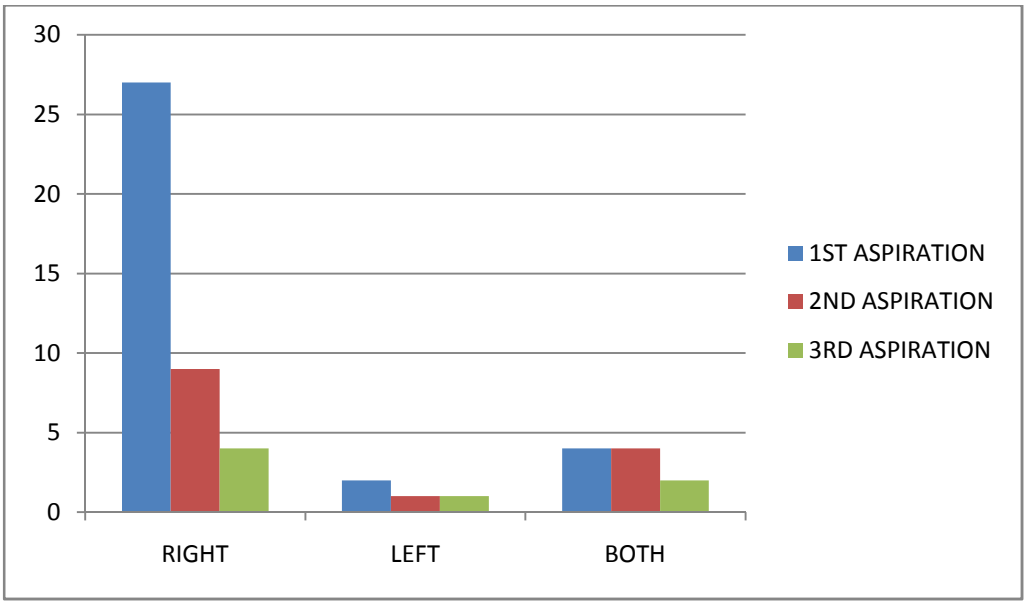


Table 6 : OUTCOME AFTER EACH ASPIRATION

S.No.	Lobe	After 1st (cure rate)	After 2nd (cure rate)	After 3rd (cure rate)
1	Right(27)	17(63.6%)	22(81%)	26(96%)
2	Left(2)	1 (50%)	1(50%)	2(100%)
3	Both(4)	0	2(50%)	4(100%)

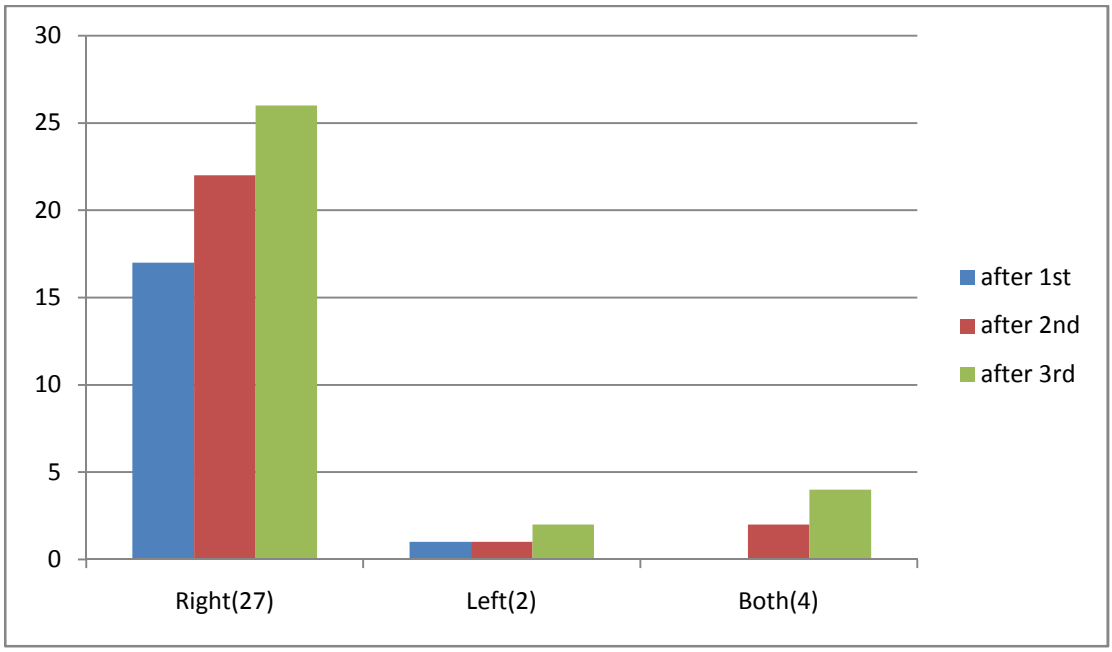


Table 7 : TUBE DRAINAGE WITH ANTIBIOTICS AND ITS OUTCOME

Sl no.	Lobe	Outcome
1	Right(2)	2(cured)
2	Left(2)	2(cured)

Table 8 : LAPAROTOMYAND DRAINAGE WITH ANTIBIOTICS

Sl no.	No. of patients	Cured	Failure
1	3	1(33.3)	2(66.6%)

Table 9 : LOCATION OF ABSCESS CAVITY AND ITS MANAGEMENT

Number of patients : 50

S.NO	LOBE	NO OF PATIENTS	PARENTERAL ANTIBIOTICS ALONE	ASPIRATION + ANTIBIOTICS	TUBE DRAINAGE + ANTIBIOTICS	LAPAROTOMY+ DRAINAGE+ ANTIBIOTICS
1	**RIGHT** SINGLE MULTIPLE	 36 05	 10 05	 24 03	 02 00	 03 00
2	**LEFT** SINGLE	 04	 01	 02	 02	 00
3	**BOTH**	04	00	04	00	00
4	**CAUDATE**	01	01	00	00	00

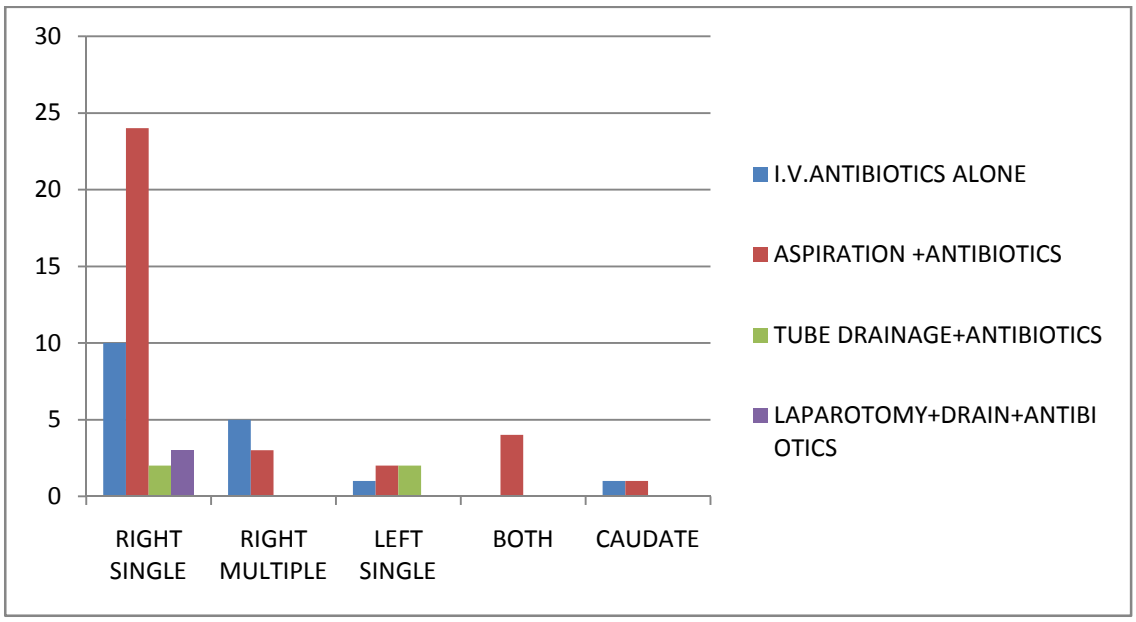


Table 10 : LOBAR INVOLVEMENT, TREATMENT AND ITS OUTCOME

Lobe involved	Mode of treatment (no)	Outcome			
		Cured		Failure	
Right lobe	Parenteral antibiotics alone-single(10)	8	80%	2	20%
	-multiple(5)	2	40%	3	60%
	After 1Aspiration	17	63%	10	37%
	2Aspiration	23	85%	4	15%
	3 Aspiration(27)	26	96%	1	4%
	Tube(2)	2	100%	0	0%
Left lobe	Antibiotics alone(1)	0	0%	1	100%
	After-1.Aspiration	1	50%	1	50%
	2.Aspiration	1	50%	1	50%
	3.Aspiration	2	100%	0	0%
	Tube(2)	2	100%	0	0%
Both lobes	Antibiotics –alone	-	-	-	-
	After-1Aspiration	0	0%	4	100%
	2Aspiration	2	50%	2	50%
	3Aspiration	4	100%	0	0%
Caudate(small abscess)	Antibiotics alone	1	100%	0	0%

DISCUSSION

Hepatic abscess was first described in ancient days by Hippocrates around 4000.B.C. (1). In 1938, Ochsner's review of 47 cases of pyogenic liver abscess were treated by open surgical drainage as the definitive therapy. Advances in diagnostic and therapeutic radiology coupled with improvements in microbiological identification and therapy have decreased the mortality rates to less than 5-30% (2).

In this series of study 50 patients were taken, among this 2 cases died.

USG and CT Scan of the abdomen were the gold standard diagnostic modalities. The choice of empirical treatment with antibiotics like ampicillin+ aminoglycosides + metronidazole or third generation cephalosporins + metronidazole were given. This regime reviewed and altered according to the culture and sensitivity after aspiration.

Malik et al (41) reported their experience of managing 169 pyogenic liver abscesses, 16 of which were treated with Intravenous antibiotics alone for 2 weeks. This approach was successful in only 6 of them, the remaining 10 required open surgical drainage, for control of sepsis.

Blessmann and colleagues reported a prospective, randomized trial of patients with amoebic abscesses that were treated with metronidazole alone or with USG – guided aspiration of the fluid plus medication. Abscess aspiration resulted in improved liver tenderness within the first 3 days, but no other difference was demonstrable between the two groups. They advocated drug treatment alone for uncomplicated abscesses with a diameter up to 10 cm and located in the right lobe of the liver (42).

In our series, 17 patients have treated with antibiotics alone. Among this patients, 11 patients were cured, 6 patients were converted into surgical methods of management. In this 11 patients, 8 patients had right lobe liver abscess with single cavity,2 patients with multiple cavity and one patient with caudate lobe abscess with small cavity.33 patients have undergone aspiration and antibiotics mode of management. After single aspiration with antibiotics, 18 patients got cured. At the end of two aspirations with antibiotics mode of treatment, 26 patients were cured. After three aspirations with antibiotics coverage, 32 patients got cured. One patient's.treatment has been changed from aspiration with antibiotics into tube drainage with antibiotics due to increasing in size of the cavity during the course of treatment. That patient presented with right lobe liver abscess with H/O pulmonary tuberculosis with completing 3 months course of anti tubercular drugs (DOTS REGIME). 32 patients were cured and 1 patient was converted into closed tube drainage.

Four patients have undergone closed tube drainage with success rate of 100%.In this category, three patients were undergone tube drainage with antibiotics directly. Indication of tube drainage due to failure of aspiration withantibiotics in pulmonary tuberculosis with right lobe liver abscess. Among this four patients, two patients were affected with right lobe liver abscess, two with left lobe abscess with large cavity with impending rupture.

Three patients were admitted with ruptured liver abscess for which laparotomy and drainage was done. Among this, 2 patients died and one patient got cured.

Patients with only left lobe liver abscess has undergone closed tube drainage in 50% of patients remaining were managed by aspiration with antibiotics.

Pus culture and sensitivity was done in all patients who had undergone aspiration or drainage procedure(open/closed).Microbial culture positive in 14% of cases, most being E.Coli. Of this study,30% of the cases had anchovy sauce pus and 34% of the cases had sterile pus. Pus culture and sensitivity was not done in 22% of patients, who were treated with I.V.Antibiotics alone.

CONCLUSION

Parenteral antibiotics alone without any drainage procedure has shown poor results in this series of studies. Only 17 patients were subjected to this mode of treatment and 6 of them needed aspiration, because of poor response to antibiotic alone. (failure rate of 36%).

Percutaneous aspiration mode of treatment has better results than I.V.Antibiotics alone treatment, with success rate of 55% after single aspiration with antibiotics in large cavity abscesses and failure cases of parenteral antibiotics alone mode of therapy, with success rate of 79% after two aspirations with antibiotics, with success rate of 97% after three aspirations with antibiotics.

Tube drainage with antibiotics mode of management was tried in failure of percutaneous aspiration with antibiotics cases and success rate of 100%.

Laparotomy and drainage with antibiotics mode of management was done only in cases, brought to the hospital with ruptured liver abscess with signs of peritonitis.The outcome of this mode was 33% of success rate.

BIBLIOGRAPHY

1. Maingot's Abdominal Operations , 11th Ed. Chapter 28. Hepatic Abscess and Cystic Disease of the Liver

2. Ochsner A. Pyogenic abscess of the liver. *Am J Surg* 1938;40:292

3. McFadzean AJS, Chang KPS, Wong CC. Solitary pyogenic abscess of the liver treated by closed aspiration and antibiotics: A report of 14 consecutive cases with recovery. *Br J Surg* 1953;41:141–152 [PubMed: 13093985]

4. Mastery of surgery, 5th Ed.

5. Sabiston Textbook of Surgery, 18th Ed.

6. Ochsner A, DeBakey M, Murray S: Pyogenic abscess of the liver. *Am J Surg* 1938; 40:292-319.

7. Couinaud C: Lobes de segments hépatiques: Notes sur l'architecture anatomique et chirurgical de foie. *Presse Méd* 62:709, 1954. [PMID: 13177441]

8. Sadler TW: *Langman's Medical Embryology*, 5th ed. Baltimore, William &Wilkins, 1985.

9. Schwartz's Principles of Surgery, 9th Ed. Chapter 31. Liver

10. GRAY'S Anatomy for students Chapter 4.Abdomen,Regional anatomy.

11. Blumgart LH, Hann LE: *Surgical and radiologic anatomy of the liver and biliary tract*. In: Blumgart LH, Fong Y, ed. *Surgery of the Liver and Biliary Tract*, London: WB Saunders; 2000:3-34.

12. Cantlie J: On a new arrangement of the right and left lobes of the liver. *Proc Anat Soc Great Britain Ireland* 32:4, 1897.

13. Bismuth H: Surgical anatomy and anatomical surgery of the liver. *World J Surg* 6:3, 1982. [PMID: 7090393]

14. Abdalla EK, Vauthey JN, Couinaud C: The caudate lobe of the liver: Implications of embryology and anatomy for surgery [review]. *Surg Oncol Clin N Am* 11:835, 2002. [PMID: 12607574]

15. Johannsen EC, Sifri CD, Madoff LC: Infections of the liver: Pyogenic liver abscess. *Infect Dis Clin N Am* 2000; 14:547-563.

16. Huang CJ, Pitt HA, Lipsett PA et al. Pyogenic hepatic abscess: Changing trends over 42 years. *Ann Surg* 1996;223:600–609 [PubMed: 8651751]

17. Pitt HA. Surgical management of hepatic abscesses. *World J Surg* 1990;14:498–504 [PubMed: 2200212]

18. Barnes S, Lillemoe K. Liver abscess and hydatid cyst disease. In: Zinner M, Schwartz S, Ellis H, Ashley S, McFadden D (eds). *Maingot's Abdominal Operations,* 10th ed. Stamford, CT: Appleton & Lange; 1997:1513–1545

20. Lambiase RE, Deyoe L, Cronan JJ, Dorfman GS. Percutaneous drainage of 335 consecutive abscesses: Results of primary drainage with 1-year follow-up. *Radiology* 1992;184:167–179 [PubMed: 1376932]

21. Kaplan GG, Gregson DB, Laupland KB: Population-based study of the epidemiology of and the risk factors for pyogenic liver abscess. *Clin Gastroenterol Hepatol* 2004; 2:1032-1038.

22. Pope IM, Poston GJ: *Pyogenic liver abscess*. In: Blumgart LH, Fong Y, ed. *Surgery of the Liver and Biliary Tract*, London: WB Saunders; 2000:1135-1145.

23. Seeto RK, Rocky DC. Pyogenic liver abscess: Changes in etiology, management, and outcome. *Medicine* 1996;75:99–113 [PubMed: 8606631]

24. Wong E, Khardori N, Carrgsco CH et al. Infectious complications of hepatic artery catheterization procedures in patients with cancer. *Rev Infect Dis* 1991;13:583–589 [PubMed: 1925273]

25. Lodhi S, Sarwari AR, Muzammil M, et al: Features distinguishing amoebic from pyogenic liver abscess: A review of 577 adult cases. *Trop Med Int Health* 2004; 9:718.

26. Hughes MA, Petri WA: Infections of the liver: Amebic liver abscess. *Infect Dis Clin N Am* 2000; 14:565-582.

27. Bailey and Love's Short practice of surgery 25th Ed.

28. Helen M Henegan, Nuala A Healy,BMC research notes, Modern management of pyogenic hepatic abscess, 4:80

29. Giorgio A, Tarantino L, Mariniello N et al. Pyogenic liver abscess: 13 years of experience in percutaneous needle aspiration with U/S guidance. *Radiology* 1995;195:122–124 [PubMed: 7892451]

30. Rajak CL, Gupta S, Jain S et al. Percutaneous treatment of liver abscesses: Needle aspiration versus catheter drainage. *Am J Roentgenol* 1998;170:1035–1039 [PubMed: 9530055]

31. Martinez Baez M. Historical introduction. In: Martinez-Palomo A (ed). *Amebiasis. Human Parasitic Diseases*, Vol. 2. Amsterdam, Holland: Elsevier; 1986:1–9

32. Tanyuksel M, Petri WA. Laboratory diagnosis of amebiasis. *Clin Microbiol Rev* 2003;16:713–729 [PubMed: 14557296].

33. Yost J. Amebiasis. *Pediatr Rev* 2002;23:293–294 [PubMed: 12154236].

34. Haque R, Huston CD, Hughes M et al. Current concepts: Amebiasis. *N Engl J Med* 2003;348:1565–1573 [PubMed: 12700377]

35.PARASITOLOGY(Protozoology and Helminthology) By K.D.Chatterjee.,THIRTEENTH EDITION.

36. Haque R, Mollah NU, Ali IKM et al. Diagnosis of amebic liver abscess and intestinal infection with the techLab *Entamoeba histolytica* II antigen detection and antibody tests. *J Clin Microbiol* 2000;38:3235–3239 [PubMed: 10970364]

37. Haque R, Huston CD, Hughes M et al. Current concepts: Amebiasis. *N Engl J Med* 2003;348:1565–1573 [PubMed: 12700377]

38. Thomas PG, Ravindra KV. Amebiasis and biliary infection. In: Blumgart LH, Fong Y (eds). *Surgery of the Liver and Biliary Tract*, 3rd ed. London, England: WB Saunders; 2001:1147–1165

39. Petri WA Jr, Singh U. Diagnosis and management of amebiasis. *Clin Infect Dis* 1999;29:1117–1125 [PubMed: 10524950]

40. Sharma MP, Dasarthy S, Verma N et al. Prognostic markers in amebic liver abscess: A prospective study. *Am J Gastroenterol* 1996;91:2584–2588 [PubMed: 8946991]

41. Malik AA, Bari SU, Rouf KA, Wani KA: Pyogenic liver abscess: Changing patterns in approach. World J Gastrointest Surg 2010, 2(12):395-401.

42. Blessmann J, Khoa ND, Van An L, et al: Ultrasound patterns and frequency of focal liver lesions after successful treatment of amebic liver abscess. *Trop Med Int Health* 2006; 11:504-508.

PROFORMA

Name: D.O.A.:

Age/Sex: D.O.S.:

I.P.No. D.O.D.

Ward:

Occupation:

Address:

Chief complaints:

1.Abdominal pain: duration,nature,site,radiation.

2.Fever: pattern,severity,duration,chills,night sweats.

3.Diarrhoea/Dysentry:duration,mucous or bloody.

4.Vomiting,anorexia

5.High coloured urine.

6.Cough.

7.Distension of Abdomen.

8.Any other.,

Past history:

H/O Similar illness.,

H/O Surgery for any illness.,

H/O DM/HTN/TB/Bronchial asthma/Epilepsy.,

H/O Trauma.,

Personal history:

Diet, sleep, bowel/bladder habit, smoking, alcohol intake,

GENERAL PHYSICAL EXAMINATION:

Nourishment

Pallor

Cyanosis

Jaundice

Clubbing

Edema

Gen. lymphadenopathy

Pulse

Blood pressure

Temperature

LOCAL EXAMINATION:

ABDOMEN:

Inspection:

1.Shape

2.Distention

3.Swelling-position,movement with respiration

4.Distended veins

Palpation:

1.Temperature/tenderness,costal tenderness

2.Extent of liver enlargement ,Consistency of swelling

3.Surface

4.Rigidiity/guarding

Percussion:

1.Upper limit liver

2.Lower limit liver below the margin

3.Fluid thrill

4.Shifting dullness

Auscultation:

Per rectal examination

RESPIRATORY SYSTEM:

Air entry

Breath sounds

CARDIOVASCULAR SYSTEM:

PROVISIONAL DIAGNOSIS

INVESTIGATIONS:

Complete haemogram

ESR

Blood urea

Serum creatinine

Blood glucose

LFT,CT/BT/PT,

Total proteins

Screening for HIV/HBsAg

Ascitic/pleural fluid analysis

Pus culture&sensitivity and for trophozoites

Urine examination

Stool analysis

Radiological investigations

X Ray chest

X Ray abdomen erect

Ultrasonogram of abdomen

Computerised tomographic scan of abdomen

CLINICAL DIAGNOSIS:

TREATMENT:

FOLLOW UP:

S.N	NAME	AGE\SEX	IP.NO	D.O.A	CXR	USG	TREATMENT	OUTCOME	REMARKS	S.N	NAME
1	Palanisamy	60\m	72927	13/12/10	NS	12*10*14 cm LEFT LOBE	5*5 cm LEFT LOBE	3.5* 2.5 cm LEFT LOBE	Tube drainage	cured	2475ml E.Coli
2	Murugesan	55\m	70604	1/12/10	Right dome of diaphragm elevated	7*4 left lobe 6.7* 6.5 right lobe	8*6.7 left lobe 8.3* 6.7 right lobe	-	Aspiration 3	cured	1400ml
3	Nagaraj	45/M	75203	25/12/10	NS	9.1*11.1*11.2cm right lobe	4.8*4.4*4.2cm right lobe	-	Aspiration 1	cured	Anchovy 200ml
4	Kannan	50/M	75980	29/12/10	Ns	10.2*10.9*13.9 cm left lobe	8.6*8.2*7.6cm left lobe	4*3.5*3.5cm left lobe	Aspiration 3	cured	990ml
5	Harikrishnan	32/M	3206	18/1/11	NS	8.9*8.4*7.8cm right lobe	3.6*3.2*2cm right lobe	-	Aspiration 1	cured	Anchovy 600ml
6	Gurusamy	45/M	4866	26/1/11	Rt. Pleural effusion	7.6*9*8.8cm right lobe	6.6*7*7.2cm right lobe	3.6*4.1*3.2 cm right lobe	Aspiration 3	cured	670 ml
7	Thangavel	57/M	6181	2/2/ 11	Rt. Pleural effusion	Right lobe 11.2*10.2*10.2 cm	6.6*5.6* 5.7cm right lobe	3.4*3.6*2.4 cm right lobe	Aspiration 3	cured	940 ml
8	Rajan	29/M	11516	1/3/11	NS	6*5.4*5.3cm right lobe	2.4*2*2.4cm right lobe	-	Aspiration 1	cured	Anchovy 250 ml
9	Murugesan	37/M	17948	3/3/ 11	Right dome elevated	Multiple- largest 3.8*2.4*2.8cm right lobe	5.4*5.6*4.8cm right lobe	-	medical	failed	Converted to aspiration. 1 280 ml
10	Nataraj	45/M	18170	1/4/ 11	NS	Multiple-largest 2*1.8*1cm right lobe	-	-	medical	cured	

11	Ammasi	65/M	22188	19/4/11	Right dome elevated	8.9*.4*9.6cm right lobe	5.8*4*4.4cm right lobe	3.2*2.4*2cm right lobe	Aspiration 2	cured	570 ml
12	Subramani	47/M	23187	22/4/11	Right dome elevated, rt. pl.effusion	7.8*8*7.8cm right lobe sub-phrenic collection	-	-	Opened &drained	cured	2100 ml
13	Swaminathan	45/M	23338	25/4/11	Right dome elevated	9.8*7.8*8.4cm right lobe	4.4*4.7*4cm right lobe	-	Aspiration 1	cured	Anchovy 540 ml
14	Muniyan	47/M	24280	30/4/11	NS	Multiple- largest 2.4*2*10cm right lobe	2*1.8*1.6cm right lobe	1.6*1.4*1.4 cm right lobe	medical	cured	-
15	Thangavel	67/M	25161	4/5/ 11	NS	4.4*4.2*3.8cm right lobe	3.6*3.4*2.8cm right lobe	2.3*2*1.8cm right lobe	medical	cured	-
16	Kamaraj	50/M	27898	17/5/11	NS	3.6*3.2*1.8cm right lobe	2.8*1.8*1.8cm right lobe	-	medical	cured	-
17	Saravanan	35/M	28535	20/5/11	Right dome elevated	11.2*10.4*9.8cm right lobe	7.8*7.4*7.6cm right lobe	2.2*2*2cm right lobe	Aspiration 2	cured	850 ml
18	Nagaraj	40\M	29449	24/5/11	Right dome elevated	11.4*11*9cm right lobe	7.8*7.4*7.8cm right lobe	4*3.6*3.4cm right lobe	Aspiration 1	Cured	Anchovy 600 ml
19	Thangavel	50\M	30272	28/5/11	Right dome elevated & rt pl.effusion	12.6*13*13.6cm right lobe	9.8*9*9.6cm right lobe	4*4.2*4cm right lobe	Tube drainage	Cured	2070 ml E.Coli
20	Ashok kumar	36\M	30977	1/6/ 11	Right dome elevated	8.8*9*9cm right lobe	6*6.5*6.2cm right lobe	3.2*2.4*2cm right lobe	Aspiration 1	Cured	Anchovy 620 ml
21	Kathirvel	44/M	34232	16/6/11	Right dome elevated	5.6*4.2*4cm right lobe	6*5.6*5cm right lobe	3*2.2*2cm right lobe	Medical	Failed	Aspiration.1 300 ml
22	Balaguru	40/M	34455	17/6/11	Rt. Pleural effusion	Ruptured liver abscess	-	-	Laparotomy and drainage	Died	1700 ml E.Coli

23	Venkatasamy	75/M	35178	21/6/11	Rt.pleural effusion	Ruptured liver abscess with peritonitis	-	-	Laparotomy & drainige	died	2200 ml
24	Appusamy	62/M	38514	7/7/ 11	NS	9.8*7.8*7.9cm right lobe	3.8*6*5.8cm right lobe	2.4*2*2.4cm right lobe	Aspiration 2	cured	950 ml
25	Sukumar	52/M	38715	8/7/ 11	NS	5.6*5*4.4cm right lobe	3.2*3*3cm	2.4*1.8*1.6	Medical	cured	-
26	Yuvaraj	37/M	40356	16/7/11	Elevated right dome	9.8*9*8.6cm Rt. lobe	7.8*7*6.4cm right lobe	4*3*2.4cm right lobe	Aspiration 1	cured	Anchovy 560 ml
27	Mani	28/M	40767	18/7/11	Normal study	3.5*2*1.8cm right lobe	1.8*1.2*1cm right lobe		Medical	cured	-
28	Thevasiappan	60/m	42204	26/7/11	Normal study	3.2*3*2 cm caudate lobe	2.4*2*2cm caudate lobe	1.6*1*0.8cm caudate lobe	Medical	cured	-
29	Murugan	45/m	42830	29/7/11	Elevated right dome	10.4*10.2*1.1 cm Right lobe	6.6*6*6 cm right lobe	3.6*3*3.2 cm right lobe	Aspiration 1	cured	780 ml
30	Babu	30/m	43733	2/8/11	Bilateral haziness	12.2*11*10.6 cm left lobe	7.4*6.8*6.8	3.5*2.8*3.0 cm left lobe	Tube drainage	cured	2150 ml
31	Rajendran	50/m	43815	3/8/11	Normal study	3.3*2.8*2.8cm right lobe	2.4*2*2 cm right lobe	1.6*1*0.8cm right lobe	Medical	cured	-
32	Chellammal	80/m	44649	7/8/11	Elevated right dome	Multiple abscess,largest rt. 3*3.5	6.6*6 cm right lobe	2.5*1.6 cm right lobe	Medical	Failed	Aspiration 3 790 ml
33	Ramasamy	32/m	45227	11/8/11	Elevated right dome	8*6.6*6.2 cm right lobe 4.8*4.6*4.6 cm eft lobe	6.6*3.5*3.6 cm right lobe 2.2*2*2.2 cm leftlobe	2.4*1.6*1.4 cm right lobe 1*0.6*0.4 cm left lobe	Aspiration 2	cured	1060 ml E.Coli

34	Balasubramani	27/m	46051	15/8/11	Elevated right dome	8.8*8.4*8.6 cm right lobe	4*4.2*4 cm right lobe	2.2*2*1.8 cm right lobe	Aspiration 1	cured	Anchovy 520 ml
35	Senthilkumar	35/m	46868	19/8/11	Elevated right dome	11*9*9.6 cm right lobe	7*5*5.2 cm right lobe	3.2*3*2.8 cm right lobe	Aspiration 1	cured	Anchovy820 ml
36	Chelladurai	55/m	47229	20/8/11	Elevated right dome	10.6*10.2*9.8 cm right lobe	6.6*6*6.2 cm right lobe	3.5*3*2.4 cm right lobe	Aspiration 1	cured	Anchovy 790 ml
37	Sivakumar	48/m	43731	22/8/11	Normal study	3.6*3*3.2 cm right lobe	2.2*1.8*1.9 cm right lobe	1.4*1*1 cm right lobe	Medical	cured	-
38	Idachiappan	32/m	47632	23/8/11	Normal study	3.2*3.3*3.2 cm right lobe	2*1.8*1.9 cm right lobe	0.8*0.6*0.6 cm right lobe	Medical	cured	-
39	Chithra	45/f	47918	24/8/11	Elevated right dome	10.9*10.4*10 cm right lobe	8.8*8.6*7.6 cm right lobe	1/*1.6*1.4 cm right lobe	Aspiration 2	cured	1280 ml
40	Kalimuthu	35/m	48341	26/8/11	Elevated right dome	5.6*4*3.6 cm right lobe 4*2.6*1.8 cm left lobe	5.6*4*3.6 cm right lobe 1.6*1*0.8 cm left lobe	2*1.4*1 cm right lobe 0.8*0.6 cm left lobe	Aspiration 2	cured	820 ml
41	Marappan	51/m	48528	27/8/11	Normal study	3.6*3.6*3.8 cm right lobe	2.4*2*2 cm right lobe	1.2-*0.8*1 cm right lobe	Medical	cured	-
42	Kumaresan	27/m	51205	10/9/11	Elevated right dome	8*7.8*7.8 cm right lobe	3.4*3.4*3.2 cm right lobe	2.2*2-*2 cm right lobe	Aspiration 1	cured	Anchovy 620 ml
43	Yaldevan	44/m	51803	13/9/11	Elevated right dome	10.8*10.6*10. 6 cm right lobe	7.6*7.6*7.4 cm right lobe	2.2*2*2 cm right lobe	Aspiration 1	cured	Anchovy 1040 ml
44	Mayilsamy	60/m	53885	23/9/11	Elevated right dome	8*7.8*7.6 cm right lobe 4*3.8*3.8 cm left lobe	5.6*5.6*5 cm right lobe 2.8*2.6*2.8 cm left lobe	2.6*2.6*2.4 cm right lobe 1*1*0.9 cm left lobe	Aspiration 3	cured	2070 ml E.Coli

45	Nagaraj	30/m	53902	23/9/11	Elevated right dome	9.8*9.6*9.6 cm right lobe	7.6*7.4*7.4 cm right lobe	3.6*3.3*3.4 cm right lobe	Aspiration 1	cured	Anchovy 630ml
46	Marutham	48/m	55362	29/9/11	Normal study	4*4.2*4 cm leftlobe	7.8*7.6*7.4 cm left lobe	1*0.9*0.8 cm right lobe	Medical	Failed	Aspiration 1 440ml
47	Arumugam	25/m	57534	3/10/11	Normal study	4*3.8*3.8*9.4 cm right lobe	9.6*9.4*9.4 cm right lobe	2.8*2.6*2 cm right lobe	Medical	Failed	Aspiration 1 780 ml
48	Saleem	32/m	58691	13/10/11	Elevated right dome	Multiple right lobe,largest 3.8*3.6*3.6	5.8*6.8*6.7 cm right lobe	2*1.8*1.9 cm right lobe	Medical	Failed	Aspiration.2 670 ml E.Coli
49	Mariappan	40/m	58908	14/10/11	Elevated right dome	8.8*9*9 cm right lobe	5.4*5.2*5.2 cm right lobe	2*1.9*1.9 cm right lobe	Aspiration 1	cured	Anchovy740 ml
50	Ramasamy(with PT)	58/m	63591	9/11/11	Elevated right dome	6*6*5.8 cm right lobe	14*10.8*10.4 cm right lobe	2.8*2.6*2.6 cm right lobe	Aspiration 1	Failed	Tube drain Anchovy 2020ml